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Signs and Symptoms of Pesticide Poisoning

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Accidental exposure or overexposure to pesticides can have serious implications. The potential for pesticide accidents is real. It has been estimated that more than 36 million pounds of pesticide active ingredients are used annually in Nebraska. While most of these pesticides can be used with relatively little risk (as long as label directions are followed), some are extremely toxic and require special precautions.

The Poison Center, at the Children's Hospital in Omaha, reports that agricultural pesticides are responsible for 4.6 percent of all accidental exposures reported. A recent study surveyed callers to The Poison Center who were exposed to agricultural chemicals. Anhydrous ammonia caused 24 percent of the incidents. The remainder were caused by herbicides (22 percent) and insecticides (54 percent). Most of the herbicide exposures resulted in eye/skin irritations. The insecticide exposures tended to result in more evident symptoms of greater concern (nausea/vomiting, headaches, dizziness, and shortness of breath).

Manage Your Risk

Wearing protective clothing and equipment when handling or applying pesticides reduces the risk of pesticide poisoning. Risk of pesticide poisoning is reduced because the chance of exposure is reduced. This idea is expressed by the Risk Formula:

Risk = Toxicity × Exposure

Understanding the toxicity of a product and the potential for personal exposure allows risk to be lowered. No matter how toxic a pesticide is, if the amount of exposure is kept low, risk can be held at an acceptably low level. The toxicity of a pesticide can't be changed, but risk can be managed by the applicator.

Signal Words

Nearly all pesticides are toxic. They differ only in the *degree* of toxicity. Because of this, pesticides are potentially dangerous to people if exposure is excessive. A pesticide product label will have one of three signal words that clearly indicate the degree of toxicity associated with that product (*Table I*). The signal words indicate the degree of potential risk to a user, not the effectiveness of the product.

Table I. pesticide label signal words and relative toxicities.

Signal Word	Toxicity	Oral Lethal Dose (Human, 150 lbs.)
Danger[a]	Highly toxic	Few drops to 1 teaspoon[b]
Warning	Moderately toxic	1 teaspoon to 1 tablespoon

Caution	Low toxicity	1 ounce to more than a pint

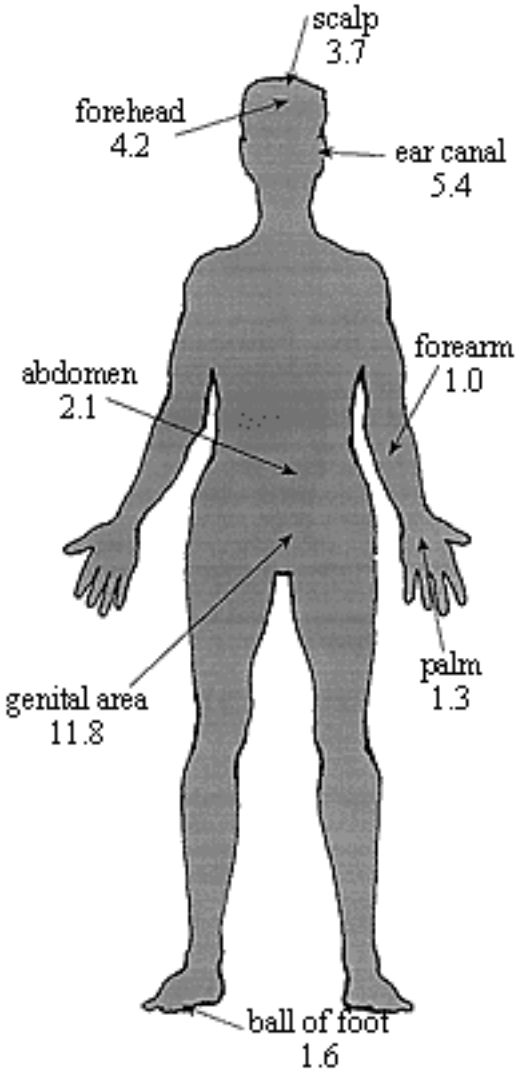
[a]The skull and crossbones symbol and the word "Poison" is often printed with the "Danger" signal word.
[b]Less for a child or person less than 150 lbs.

Read the Pesticide Label

Along with the signal words, pesticide labels also include statements about route of entry and specific actions that must be taken to avoid exposure. Route of entry statements indicate the outcome that can be expected from exposure. For example, a pesticide label might read: "Poisonous if swallowed, inhaled, or absorbed through the skin. Rapidly absorbed through the skin and eyes." This indicates that the pesticide is a potential hazard through all three routes of entry, and that skin and eye contact are particularly hazardous. Specific action statements normally follow the route of entry statement and indicate what must be done to prevent poisoning accidents. In the case of the pesticide discussed above, the statement might read: *"Do not get in eyes, on skin, or on clothing. Do not breathe spray mist."*

Routes of Exposure

pesticides can enter the human body three ways: 1) by absorption through the skin or eyes (***dermally***); 2) through the mouth (***orally***); and 3) by breathing into the lungs (***inhalation***).



Dermal exposure results in absorption immediately after a pesticide contacts skin or eyes. Absorption will continue as long as the pesticide remains in contact with the skin. The *rate* at which dermal absorption occurs is different for each part of the body (*Figure 1*). The relative absorption rates are determined by comparing each respective absorption rate with the forearm absorption rate.

Figure 1. Relative absorption rates, compared to the forearm with an absorption rate of 1.0.

It is easy to transfer pesticide residues from one part of the body to another. When this occurs, the applicator increases the potential for pesticide poisoning. For example, residues can be inadvertently moved from a hand to a sweaty forehead (4.2) or to the genital area (11.8). At this very high rate, the absorption of a pesticide is more dangerous than swallowing it!

Oral exposure may result in serious illness, severe injury, or even death, if a pesticide is swallowed. pesticides can be ingested by accident, through carelessness, or intentionally.

The most common accidental oral exposures occur when pesticides have been removed from their original containers and placed into an unlabeled bottle, jar or food container. Children under 10 are victims of at least 1/2 of the accidental

pesticide deaths in the United States. If pesticides were managed properly, children would never touch them.

Follow these guidelines:

- Always store pesticides in their original labeled containers.
- Never use the mouth to clear a spray hose or nozzle, or to begin siphoning a pesticide.
- Never eat, drink or use tobacco until after leaving the work area and washing thoroughly.

Respiratory exposure is particularly hazardous because pesticide particles can be rapidly absorbed by the lungs into the bloodstream. pesticides can cause serious damage to nose, throat, and lung tissue if inhaled in sufficient amounts. Vapors and very small particles pose the most serious risks.

Lungs can be exposed to pesticides by inhalation of powders, airborne droplets or vapors. Handling concentrated wettable powders can pose a hazard if inhaled during mixing. The hazard from inhaling pesticide spray droplets is fairly low when dilute sprays are applied with low pressure application equipment. This is because most droplets are too large to remain airborne long enough to be inhaled.

However, when high pressure, ultra low volume (ULV), or fogging equipment is used, the potential for respiratory exposure is increased. The droplets produced during these operations are in the mist- or fog-size range and can be carried on air currents for a considerable distance.

Pesticide Toxicity

The toxicity of a pesticide can be measured several ways. Determining the toxicity of pesticides to humans is not easy. Obviously humans can't be test animals. Other animals, usually rats, are used. However, if a pesticide is poisonous to rats, it is not necessarily poisonous to dogs, cows, wildlife or people. Toxicity studies are only guidelines. They are used to estimate how poisonous one pesticide is compared to another pesticide. Some pesticides are dangerous after one large dose (exposure). Others can be dangerous after small, repeated doses.

Measuring toxicity. The toxicity of a pesticide is determined by laboratory testing on animals such as rats, mice and rabbits. The measuring method, LD_{50} (lethal dose, 50 percent), describes the dose of a pesticide that will kill half of a group of test animals from a single exposure (dose) by either the dermal, oral or inhalation routes. A pesticide with a lower LD_{50} is more toxic than a pesticide with a higher number because it takes less of the pesticide to kill half of the test animals.

The toxicity of fumigant pesticides is described in terms of the concentration of the pesticide in the air, LC_{50} (lethal concentration, 50 percent). A similar system is used to test the potential effects of pesticides against aquatic organisms in water.

Acute toxicity of a pesticide refers to the effects from a single exposure or repeated exposure over a short time, such as an accident during mixing or applying pesticides. Various signs and symptoms are associated with acute poisonings.

A pesticide with a high acute toxicity can be deadly even if a small amount is absorbed. It can be measured as acute oral toxicity, acute dermal toxicity or acute inhalation toxicity.

Chronic toxicity refers to the effects of long-term or repeated lower level exposures to a toxic substance.

The effects of chronic exposure do not appear immediately after first exposure and may take years to produce signs and symptoms. Examples of chronic poisoning effects may include:

- Carcinogenicity--ability to produce cancer or to assist carcinogenic chemicals.
- Mutagenicity--ability to cause genetic changes.
- Teratogenicity--ability to cause birth defects.
- Oncogenicity--ability to induce tumor growth (not necessarily cancers).
- Liver damage--death of liver cells, jaundice (yellowing of the skin), fibrosis and cirrhosis.
- Reproductive disorders--such as reduced sperm count, sterility, and miscarriage.
- Nerve damage--including accumulative effects on cholinesterase depression associated with organophosphate insecticides.
- Allergenic sensitization--development of allergies to pesticides or chemicals used in formulation of pesticides.

The effects of chronic toxicity, as with acute toxicity, are dose-related. In other words, low-level exposure to chemicals that have potential to cause long-term effects may not cause immediate injury, but repeated exposures through careless handling or misuse can greatly increase the risk of chronic adverse effects.

Recognizing Signs and Symptoms of Poisoning

Anyone who may become exposed to pesticides should be aware of the signs and symptoms of pesticide poisoning. Prompt action during pesticide overexposure can prevent serious consequences.

Poisoning signs can be seen by others, for example, vomiting, sweating, or pin-point pupils. Symptoms are any functional changes in normal condition which can be described by the victim of poisoning, and may include nausea, headache, weakness, dizziness, and others. Anyone who works with pesticides should learn what these signs and symptoms are to prevent serious injury and allow prompt treatment.

Persons who are frequently involved with pesticides should become familiar with these important steps:

1. Recognize the signs and symptoms of pesticide poisoning for those pesticides you commonly use or to which you may be exposed.
2. If you suspect a pesticide poisoning, get immediate help from a local hospital, physician, or the nearest poison control center.
3. In a pesticide emergency, identify the pesticide to which the victim was exposed. Provide this information to medical authorities.
4. Have a copy of the pesticide label present when medical attention is begun. The label provides information that will be useful in assisting a pesticide poisoning victim.
5. Know emergency measures you can take until help arrives or the victim can be taken to the hospital. Both first aid and medical treatment procedures are listed on the product label.

Recognizing Common Pesticide Poisonings

All pesticides in a given chemical group generally affect the human body in the same way; however, severity of the effects vary depending on the formulation, concentration, toxicity and route of exposure of the pesticide. It is important, therefore, to know both the type of pesticide you are using and the signs and symptoms associated with poisoning from it.

Organophosphate and Carbamate Insecticides

Most pesticide poisoning cases involve either organophosphate or carbamate insecticides. Both chemical groups affect humans by inhibiting acetyl cholinesterase, an enzyme essential to proper functioning of the nervous system. Some organophosphate and carbamate insecticides commonly used in Nebraska are listed in *Table II*.

Table II. Common organophosphate and carbamate insecticides.

Organophosphates	
Actellic	Lorsban, Dursban
Co-Ral	Malathion
Counter	Metasystox (I), R or S
Cygon	Methyl-parathion,
Dasanit	Ethyl-parathion
Delnav	Mocap
Diazinon, Spectracide	Oftanol
Dibrom	Orthene
Di-Syston	Penncap-M
Dyfonate	Phosdrin
Dylox	Supracide
EPN	Thimet
Guthion	Vapona, DDVP
Imidan	
Carbamates	
Baygon	Nudrin
Broot	Sevin, carbaryl
Furadan	Temik
Lannate	

The effects of these materials, particularly organophosphate insecticides, are rapid. Symptoms begin shortly after exposure, and in acute poisonings, during the exposure. Exposure to either of these insecticide classes may pose special risks for persons with reduced lung function, convulsive disorders, etc. In some cases, alcoholic beverage consumption may exacerbate the pesticide effects.

The onset of symptoms in milder exposures can occur anytime up to 12 hours later, but usually within four hours. Consequently, diagnosis of a suspected poisoning must also be rapid. **It is imperative to be familiar with the signs and symptoms these types of pesticides cause.**

Signs and symptoms associated with **mild** exposures to organophosphate and carbamate insecticides include:

- headache, fatigue, dizziness, loss of appetite with nausea, stomach cramps and diarrhea;
- blurred vision associated with excessive tearing;
- contracted pupils of the eye;
- excessive sweating and salivation;
- slowed heartbeat, often fewer than 50 per minute;
- rippling of surface muscles just under the skin.

These symptoms may be mistaken for those of flu, heat stroke or heat exhaustion, or upset stomach.

Moderately severe organophosphate and carbamate insecticide poisoning cases exhibit all the signs and symptoms found in mild poisonings, but in addition, the victim:

- is unable to walk;
- often complains of chest discomfort and tightness;
- exhibits marked constriction of the pupils (pinpoint pupils);
- exhibits muscle twitching;
- has involuntary urination and bowel movement.

Severe poisonings are indicated by incontinence, unconsciousness and seizures.

The order in which these symptoms appear may vary, depending on how contact is made with the pesticide. If the product is swallowed, stomach and other abdominal manifestations commonly appear first; if it is absorbed through the skin, gastric and respiratory symptoms tend to appear at the same time.

Fortunately, good antidotes are available for victims of organophosphate or carbamate poisoning at emergency treatment centers, hospitals, and many physicians' offices. **As with all pesticide poisonings, time is extremely critical.** If a pesticide is swallowed, obtain prompt medical treatment. If a dermal exposure has occurred, remove contaminated clothing, wash exposed skin and seek medical care.

Organochlorine Insecticides

The U. S. Environmental Protection Agency has sharply curtailed the availability of many organochlorines because they are not readily biodegradable and persist in the environment. These materials affect the nervous system as stimulants or convulsants. Two organochlorine insecticides, lindane and methoxychlor, still have limited use.

Nausea and vomiting commonly occur soon after ingesting organochlorines. Other early signs and symptoms include: apprehension, excitability, dizziness, headache, disorientation, weakness, a tingling or pricking sensation on the skin, and muscle twitching. This is followed by loss of coordination, convulsions similar to epileptic seizures, and unconsciousness. When chemicals are absorbed through the skin, apprehension, twitching, tremors, confusion, and convulsions may be the first symptoms.

No specific antidotes are available for organochlorine poisoning. Remove contaminated clothing immediately, and then bathe and shampoo the person vigorously with soap and water to remove pesticide from the skin and hair. Persons assisting a victim should wear chemical resistant gloves and be careful to avoid becoming contaminated by the pesticide. If the pesticide has been ingested, empty the stomach as soon as possible by giving the conscious patient ipecac and water or by inserting a finger into the throat.

Caution: Inhaling vomitus can be life-threatening. Timely emergency treatment is vital to survival.

Synthetic Pyrethroid Insecticides

Pyrethroids are synthetically produced compounds that mimic the structure of naturally occurring pyrethrins. Some examples of pyrethroids are in *Table III*.

Systemic toxicity by inhalation and dermal absorption is low. There have been very few systemic poisonings of humans by pyrethroids. Dermal contact may result in skin irritation such as stinging, burning, itching, and tingling progressing to numbness.

Table III. Commonly used synthetic pyrethroid insecticides.

- Allethrin (Pynamin)
- Cyfluthrin (Baythroid)
- Cypermethrin (Ammo, Cynoff, Cymbush)
- Esfenvalerate (Asana)
- Fenvalerate (Pydrin)
- Flucythrinate (Pay-off)
- Fluvalinate (Mavrik)
- Permethrin (Ambush, Dragnet, Pounce, Torpedo)
- Resmethrin (Outdoor Insect Fogger, Scourge, Synthrin)
- Tetramethrin (Flea Killer II)
- Tralomethrin (Scout)

Some may be toxic by the oral route, but usually ingestion of pyrethroid insecticide presents relatively little risk. Very large doses may rarely cause incoordination, tremors, salivation, vomiting, diarrhea, and irritability to sound and touch. Most pyrethroid metabolites are promptly excreted by the kidney. Pyrethroids are not cholinesterase inhibitors.

Plant-derived Insecticides

Pyrethrum and pyrethrins. Crude pyrethrum is a dermal and respiratory allergen. Skin irritation and asthma have occurred following exposures. The refined pyrethrins are less allergenic, but appear to retain some irritant and/or sensitizing properties.

In cases of human exposure to commercial products, the possible role of other toxicants in the products should be considered. The synergists, such as piperonyl butoxide, have low toxic potential in humans, but organophosphates or carbamates included in the product may have significant toxicity. Pyrethrins themselves do not inhibit the cholinesterase enzyme.

Rotenone. This naturally occurring substance is present in many plants. It is formulated as dusts, powders, and sprays for use in gardens and on food crops. Although rotenone is toxic to the nervous systems of insects, fish, and birds, commercial rotenone products have presented little hazard to humans.

Inorganic Insecticides

Boric Acid and Borates. Borax dust is moderately irritating to skin. Inhaled dust causes irritation of the respiratory tract and shortness of breath.

In severe poisonings of infants, a beefy red skin rash, most often affecting palms, soles, buttocks, and scrotum, has been described. It has been characterized as a "boiled lobster appearance." The intense redness of the skin is followed by extensive skin peeling.

Microbial Insecticides

Bacillus thuringiensis (Bt). From studies involving deliberate ingestion by human subjects, it appears possible that the organism can cause inflammation of the digestive tract. No irritation or sensitization effects have been reported in workers preparing and applying commercial products.

DEET Repellent

DEET (Detamide, MGK, OFF). For many years, diethyltoluamide has been effective and generally well tolerated when applied to human skin, although tingling and mild irritation have followed repeated application. In some cases, DEET has caused skin irritation and intensification of preexisting skin disease. It is very irritating to the eyes, but not corrosive.

Serious adverse effects have occurred when the product has been used under hot, humid conditions and applied to skin areas that are in direct contact during sleep. Under these conditions, the skin became red and tender, then exhibited blistering and erosion, leaving painful weeping bare areas that were slow to heal. Permanent scarring resulted from most of these severe reactions.

Great caution should be exercised in using DEET on children. Only the products containing the lower concentrations should be used, and application should be limited to clothing, using as little repellent as possible. If headache or any kind of emotional or behavioral change occurs, use of DEET should be discontinued immediately.

Table IV. Herbicides with similar signs and symptoms.

These herbicides share common signs and symptoms. In general, these herbicides can irritate the skin, eyes, and respiratory tract. They exhibit a low systemic toxicity.

Herbicide Class	Common Name	Trade Name
Dinitroanilines	Pendimethalin	Prowl, Stomp
	Oryzalin	Surflan
Nitriles	Bromoxynil	Buctril, Merit
Aliphatic acids	Dalapon	Dalacide, Unipon
Phenoxy-benzoic acids	Dicamba	Banvel
Phosphonates	Glyphosate	Roundup, Range, Rodeo, Kleenup
Pyridines	Picloram	Tordon
	Triclopyr	Garlon, Grandstand, Turflon
Substituted amides	Alachor	Lasso, Partner
	Metolachlor	Dual, Dual II, Pennant

	Propanil	Stampede, Surcopur
	Pronamide	Kerb
Thiocarbamates	Butylate	Sutan+
	EPTC	Eptam, Eradicane
	Molinate	Ordram
	Triallate	Buckle, Far-Go
Triazine and Triazoles	Amitrol	Amitrol, Amizol
	Atrazine	AAtrex, Atrazine
	Cyanazine	Bladex, Fortrol
	Hexazinone	Velpar
	Metribuzin	Sencor, Sencoral, Sencorex
	Propazine	Primatol
	Simazine	Princep, Caliper 90
Uracils	Bromacil	Hyvar
	terbacil	Sinbar
Ureas	Diuron	Seduron, Direx, Karmex
	fluometuron	Cotoran
	Linuron	Linex, Lorox

Bipyridyl Herbicides

The most common bipyridyls are diquat and paraquat. Paraquat is more toxic than diquat and produces chronic abnormal cell growth in the lungs, cornea and lens of the eye, nasal mucosa, skin, and fingernails. Diquat affects the eye lens and intestinal tract lining, but does not usually produce the frequently fatal lung changes characteristic of paraquat.

Ingesting diquat or paraquat causes severe irritation to the mucous membranes of the mouth, esophagus, and stomach. Repeated vomiting generally follows. Large doses of diquat also produce restlessness and reduced sensitivity to stimulation.

Large doses (6 to 8 oz.) of paraquat initially can affect the lungs, kidneys, liver and adrenals; potentially fatal fluid accumulation in the lungs can occur in 24 to 72 hours.

Lesser amounts of paraquat will cause decreased urine volume in one to six days because of kidney failure; yellowing of the skin (jaundice) due to liver damage sometimes is observed. The initial phase is followed by a latent period lasting up to two weeks, during which the victim appears to improve. However, irreversible and progressive lung damage caused by rapid growth of connective tissue cells that prevent proper lung function eventually leads to death through respiratory failure. Paraquat selectively concentrates in pulmonary cells.

Dermal exposure to paraquat and diquat concentrates may cause severe skin irritation and burning. Contact with dilute liquids and diquat dusts may cause slight to moderate irritation. Dermal absorption of paraquat apparently is slight, but diquat is absorbed and after repeated contact will produce symptoms similar to those following ingestion.

Exposure to paraquat and diquat spray mist may produce skin irritations, nasal bleeding, irritation and inflammation of the mouth and upper respiratory tract, coughing and chest pain. Exposure to paraquat concentrates may cause blackening of the nails and abnormal nail growth.

There are no specific antidotes to counteract effects of paraquat and other bipyridyl herbicides once significant exposure and absorption has occurred. If ingested, induce vomiting immediately if not contraindicated by physical condition. Flush affected eyes with water, or wash skin with soap and water. Seek medical attention promptly.

Chlorophenoxy Herbicides

Compounds such as 2,4-D and MCPA are examples of chlorophenoxy herbicides. These compounds are moderately irritating to skin and mucous membranes. Inhalations may cause burning sensations in the nose, sinuses and chest, and coughing may result. Prolonged inhalation sometimes causes dizziness.

Irritation of the stomach usually leads to vomiting soon after ingestion. Chest and abdomen pain and diarrhea may ensue. Headache, mental confusion, and bizarre behavior are early signs and symptoms of severe poisoning which may progress to unconsciousness.

Arsenical Herbicides

Examples include Ansar and Montar. **Acute arsenic poisoning** usually appears within one hour of ingestion. Garlic odor of the breath and feces may help to identify the responsible toxicant in severe cases. Digestive tract effects include inflammation of the mouth and esophagus, burning abdominal pain, thirst, vomiting, and "ricewater" or bloody diarrhea.

Central nervous system effects include headache, dizziness, muscle weakness and spasms, low body temperature, sluggishness, delirium, coma, and convulsions. Liver damage may lead to yellowness of the skin. Injury to blood-forming tissues may cause a reduction in red and white blood cells and blood platelets. Death usually occurs one to three days after symptom onset and is usually the result of circulatory failure.

Chronic arsenic poisoning. Dermal manifestations are usually more prominent than the intestinal tract effects which characterize acute poisoning: overgrowth of the cornea or epidermis; scaling off of dead skin; excessive fluids under the skin of the face, eyelids, and ankles; white streaks across the nails; loss of nails or hair; and brick red coloration of visible mucus membranes.

Wood Preservatives

Creosote (coal tar) exposure can cause skin irritation and prolonged exposure may lead to dermatitis. Vapors and fumes of creosote are irritating to the eyes and respiratory tract. Ingested creosote may result in severe liver damage.

Pentachlorophenol (PCP, Penchlorol, Penta) is irritating to the eyes, skin, and respiratory tract, causing stuffy nose, scratchy throat, and tearing. Prolonged dermal exposure sometimes leads to an acne-like skin condition. Ingestion of PCP solutions, excessive skin contact, or inhalation of concentrated vapors may cause fever, headache, weakness, dizziness, nausea, and profuse sweating. Extreme cases can induce loss of coordination and convulsions, high fever, muscle spasms and tremors, labored breathing, a sense of constriction in the chest, abdominal pain and vomiting, restlessness, excitement, and mental confusion. Intense thirst is also characteristic. Pentachlorophenol poisoning can be fatal, having caused cardiac and muscular collapse.

Arsenical wood preservatives (chromated copper arsenate [CCA] and ammoniacal copper arsenate [ACA]) can cause nausea, headache, diarrhea, and abdominal pain (if swallowed). Extreme signs and symptoms can progress to dizziness, muscle spasms, delirium and convulsion. Prolonged exposure to arsenical wood preservatives can result in persistent headache, abdominal distress, salivation, low grade fever, and upper respiratory irritation.

Fumigants

Chloropicrin, methyl bromide, sulfuryl fluoride (Vikane) and phosphine (generated by aluminum or magnesium phosphide, e.g., Phostoxin, Fumitoxin and Fumi-Cel) are commercial fumigant products.

Various types of fumigants produce differing physiologic effects. Headache, dizziness, nausea and vomiting are common early signs and symptoms of excessive exposure.

Sulfuryl fluoride poisoning symptoms include depression, slowed gait, slurred speech, nausea, vomiting, stomach pain, drunkenness, itching, numbness, twitching, and seizures. Inhalation may be fatal due to respiratory failure. Inhalation of high concentrations may cause respiratory tract irritation. Skin contact with sulfuryl fluoride normally poses no hazard, but contact with liquid sulfuryl fluoride can cause pain and frostbite due to rapid evaporation.

Phosphine fumigants, such as aluminum and magnesium phosphide, affect cell function, liver and lungs. ***Mild exposure*** is signaled by a sensation of cold, chest pains, diarrhea and vomiting. Somewhat more serious exposures will be evidenced by cough, chest tightness, difficult breathing, weakness, thirst and anxiety.

Severe exposure is indicated by stomach pain, loss of coordination, blue skin color, limb pain, enlarged pupils, choking, fluid in the lungs and stupor. Severe poisonings lead to seizures, coma and death.

Halocarbon fumigants (e.g. chloropicrin and methyl bromide) affect the central nervous systems, lungs, heart and liver. Persons poisoned by this type of fumigant experience the common signs and symptoms of fumigant poisoning along with abdominal pain, weakness, slurred speech, mental confusion, tremors, and convulsions similar to epileptic seizures. Some liquid fumigants cause skin injuries indicated by areas of redness or blisters that rupture, leaving raw skin or deep ulcers.

Move victims of fumigant inhalation to fresh air immediately. Keep the individual quiet in a semi-reclining position even though initial signs and symptoms are mild. If breathing has stopped, give mouth-to-mouth or mouth-to-nose resuscitation. If there is no pulse, use cardiopulmonary resuscitation (CPR). Time is particularly critical in fumigant poisonings; victims must be given prompt medical attention.

Rodenticides

Coumarins. Examples include brodifacoum (Havoc, Talon, WeatherBlok), bromadiolone (Contrac, Maki), and warfarin. Intestinal tract absorption of these toxicants is efficient. The main signs and symptoms are nosebleed, bleeding gums, blood in the urine, tar feces, and large irregular blue-black to greenish-brown spots or patches on the skin. There is concern that the more toxic modern compounds, such as brodifacoum, may cause serious poisoning of nontarget mammals, including humans, at much lower dosages.

Indandiones. Examples are chlorophacinone (Rozol) and diphacinone (Diphacin, Ramik). Unlike the coumarin compounds, some indandiones cause signs and symptoms of nerve, heart and circulatory system damage in laboratory rats leading to death before hemorrhage occurs. None of these signs and symptoms have been reported in human poisonings.

Zinc phosphide. Zinc phosphide causes severe irritation if ingested. It reacts with water and stomach juices to release phosphine gas which can enter the blood stream and affect the lungs, liver, kidneys, heart and central nervous system. Zinc phosphide is easily absorbed through the skin or inhaled from fumes. With repeated exposure, it accumulates in the body to dangerous levels.

Signs and symptoms of mild zinc phosphide poisoning include diarrhea and stomach pains. In more severe cases, nausea, vomiting, chest tightness, excitement, coldness, unconsciousness, coma and death can occur from pulmonary edema and liver damage. There is no antidote for zinc phosphide poisoning. It is a slow-acting material, which gives the victim time to get medical help.

Strychnine. Strychnine is not easily absorbed through the skin nor does it accumulate in the human body. When ingested, it acts on the central nervous system within 10 to 30 minutes. Violent convulsions occur, causing breathing to stop.

Treatment of strychnine poisoning is geared toward eliminating outside stimuli. If strychnine poisoning occurs, it is important to place the victim in a warm, dark room, which reduces outside stimuli that trigger convulsions. Consequently, **in the case of strychnine poisoning, bring medical help to the victim rather than transporting the victim** to a medical center, because movement will trigger convulsions.

Fungicides

Fungicides are extensively used in industry, agriculture, the home and garden. Fungicides vary enormously in their potential for causing adverse effects in humans. Most fungicides currently in use are unlikely to cause frequent or severe poisonings. Apart from poisonings that affect the body generally, fungicides have probably caused disproportionate numbers of irritant injuries to skin and mucous membranes, as well as some dermal sensitization. *Table V* lists several fungicides divided by class.

Table V. Fungicides listed by class with common and trade names.

Fungicide Class	Common Name	Trade Name
Acylalanine	Metalaxyl	Ridomil, Apron
Analide	Carboxin	Oxatin, Vitavax
Benzimidazole	Benomyl	Funomyl, Benex, Benlate
	Thiabendazole	Arbotect, Mertect, Storite
Substituted Aromatics	Chlorothalonil	Bravo, Daconil 2787, Tuffcide
	Hexachlorobenzene (HCB)	No Bunt
	PCNB (Quintozene)	Terraclor, Turcide
Dicarboximide	Iprodione	Kidan, Rovral, Chipco 26019
	Vinclozolin	Ronilan, Ornalin
Dinitrophenyl	Dinocap	Crotothane, Mildane
Dithiocarbamates	Mancozeb	Dithane, Fore, Tridex, Manzate
	Maneb	Manex, Manox, Maneb 80

	Thiram	Thiram 75 WG, Tripomol
Guanidine	Dodine	Venturol, Melprex
Inorganic	Copper Sulfate	Blue Viking, Triangle Brand
Phthalimide	Captan	Captanex, Merpan

What To Do When Pesticide Poisoning Occurs

The key to surviving and recovering from a pesticide poisoning is ***rapid*** treatment. **Take emergency action immediately when you suspect a pesticide poisoning.** As time continues to elapse after exposure, recovery is hindered and the toxic effects are heightened.

If the common emergency telephone number is available in your area, immediately call **911** whenever a pesticide poisoning is suspected. An advanced life support team will be dispatched to provide assistance.

If the common emergency telephone number is ***not*** available in your community, contact:

1. **The Poison Center, 1-800-955-9119.** The poison center will be able to provide specific directions on procedures to follow until a life support team arrives,
2. the nearest hospital,
3. a physician.

Another source of medical information related to pesticides during non-emergencies is the National Pesticide Telecommunications Network, **1-800-858-7378.** Medical and consumer information on pesticides is available through this hotline.

What a victim might think is a cold or the flu could be a fatal pesticide poisoning. Whenever possible, find out the following critical information:

1. Has the victim been exposed to a pesticide?
2. If so, which one and how did the exposure occur?
3. What emergency actions are on the pesticide label?

Many labels direct that vomiting be induced. Vomiting can be induced by giving the patient ipecac and water or by inserting the finger into the throat of the victim. **Do not induce vomiting when:**

1. the label says not to;
2. convulsions have occurred;
3. the victim is unconscious; and
4. the pesticide contains petroleum products such as xylene.

Always wash the victim's exposed skin with a detergent and plenty of water. Skin irritation can result from continuous exposure if not treated. If skin exposure occurs, obtain medical treatment. If the victim's clothing has been contaminated by a pesticide that is readily absorbed dermally, remove the clothing and decontaminate the victim's skin.

Even though careful pesticide application is normal, accidents can happen. Be prepared. Keep the number for the Poison Center readily available either in your telephone directory or near your telephone. Do not hesitate to contact medical authorities if any symptoms of pesticide poisoning occur. It is better to

be safe than sorry.

Most pesticides used by Nebraska farmers and ranchers, lawn owners, and gardeners exhibit lower toxicity than most pesticides discussed in this publication. When applied properly, with the required protective clothing and equipment, they are unlikely to cause problems for the user.

However, ***any*** pesticide ***can*** cause exposure problems. **Use all pesticides safely. Read the pesticide label completely and comply with all directions. Failure to do so may subject you to sanctions or penalties provided by federal and/or state laws.**

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Handbook of pesticide Toxicology. 1991. Wayland J. Hayes Jr. and Edward R. Laws Jr. Editors. Academic Press, Inc. San Diego, CA.

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Recognition and Management of pesticide Poisonings. Donald P. Morgan. Fourth Edition, 1989. Document No. 055-000-00359-9. Supt. of Documents, U.S. Government Printing Office, Washington, DC 20402-9325.

pesticide Safety Telephone Numbers

Nonemergency Telephone Numbers	
National pesticide Telecommunication Network	**(800) 858-7378**
Chemical Referral Center (weekdays only)	**(800) 262-8200**
Referrals to manufacturers on health and safety related to chemicals	
Emergency Telephone Numbers	
The Poison Center	**(800) 955-9119**
For aid in human poisoning cases	
Pesticide Accident Hotline (CHEMTREC)	**(800) 424-9300**
For help involving spills, leaks, fires	
Nebraska State Patrol	**(800) 525-5555**
To report chemical spills or releases	
To report motor vehicle accidents	

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